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Children's Blood Lead Levels in New Haven: A Population-Based Demographic Profile

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This report presents the geometric mean blood lead levels of an 80% cross-sectional sample of children aged 1-72 months in New Haven, Connecticut. Blood lead levels were related to age, sex and race. It was found that age and race were independently important sources of variation in blood lead levels. Sex of children in this age group was not related to differences in blood lead levels. The highest geometric mean blood lead levels occurred in children between 25 and 36 months of age. Black children had higher levels than white or Hispanic children.

Introduction

In the extensive literature on childhood lead poisoning there is little descriptive information about blood lead levels among urban children divided by age, sex and race. Numerous studies have focused on children at highest risk or children with undue lead absorption. Indeed, Chisolm has stated that there are, as yet, no studies designed to measure incidence on the basis of sound epidemiological principles (*1*).

Several estimates of blood lead in U.S. children have been published. They vary widely. Gilsinn (*2*) derived national estimates by using data from the 1970 census for housing built before 1950 and blood lead data from eastern and midwestern large urban areas. She estimated a nationwide rate of 28.9% of children with blood lead concentrations $\geq$ 40 μg/dl, 12.7% $\geq$ 50 μg/dl, 6% $\geq$ 60 μg/dl, and 2.7% $\geq$ 70 μg/dl. In contrast, a recent Center for Disease Control report (*3*) stated that since 1972 lead poisoning prevention programs located in all sections of the country have reported the screening of 3,350,000 children, ages 1-5; 221,000 (6.6%) have been identified as having blood lead levels $\geq$ 30 μg/dl.

A number of factors have been identified as influencing the likelihood of an increased body burden of lead. These include age, race, socioeconomic status, housing and season. Sex does not seem to be an important factor (*4*, *5*). Children from one through six are at risk for childhood lead poisoning, with peak incidence occurring between one and three years (*6*).

It is not clear whether race acts independently or merely in association with other factors. Many studies have looked only at high risk populations where racial distribution is highly imbalanced. Jacobnizer (*7*) reported a disproportionately high incidence of elevated blood lead levels in the Puerto Rican and nonwhite groups in a high risk neighborhood screening program. Such neighborhoods are usually characterized by slums and dilapidated housing and children living there are generally at

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greater risk for increased lead absorption than are those in better quality neighborhoods (*7, 8*). Incidence of cases of acute childhood lead poisoning peaks in the summer months (May-October) for a mixture of reasons, including sunshine, dietary calcium, vitamin D, dehydration and availability of lead from outdoor paint, soil and fallout from the air, as well as other factors not yet understood (*3, 8-10*).

Methods

The Health Department of the City of New Haven carried out a screening program from July 1974 to February 1977. All children residing within the city who were 1 through 72 months old were eligible for screening. A variety of publicity approaches were utilized to inform parents about the screening program and to urge participation. The Health Department provided the screening test free of charge at a number of locations throughout the city, including in a mobile van and in the childs' own home, if desired. Parents who chose not to use these services were encouraged to have their children tested by their own physicians. All blood lead tests in New Haven, regardless of who performs them, are required to be reported to the Health Department.

The purpose of the program was explained to the parent(s) or guardian of each child to be included. Written consent was obtained from parent or guardian before each test was performed. Detailed information was obtained on a cross section of the population of interest (*11*). This study group was composed of 7912 children in the age group 1 through 72 months of age at the time of test and represented about 80% of the entire 1-72 month old resident pediatric population during this time period. Of the remaining 20%, approximately 1% comprised children who were screened but not included in this report because their racial designation was other than black, white or Hispanic. The other 19% were not proportionally distributed and tended to be of higher socioeconomic status and to be white. This resulted from the less intense effort to screen children thought not to be at risk for lead poisoning. For each child in the sample a blood lead measurement was obtained.

All sample collections by the New Haven Health Department were done by either of two individuals, a certified laboratory technician or a registered nurse who had been trained by the technician. The sampling technique was standardized as follows. The child's middle finger was thoroughly cleansed with soap solution, rinsed and dried. The finger was then sprayed with collodion, which was allowed to dry. A disposable lancet was used to puncture the finger from which approximately ten drops of blood were allowed to drip onto the filter paper which was then inserted into a plastic envelope. The envelope was sealed and refrigerated for a maximum of 24 hr before being mailed to the State Health Department Laboratory. Collections by other medical care providers were done by nurses, physicians or trained technicians. Samples were collected in individual homes as well as clinics, hospitals and private care facilities. With the exception of Yale-New Haven Hospital, all medical care providers sent their blood samples to the Connecticut State Health Department Laboratory. The State used the Dithizone method (*12*) of analysis for macro (5-10 ml) samples and atomic absorption spectrophotometry with Delves' cup attachment (*13*) for micro samples. The State Laboratory has been cooperating with the Childhood Lead Poisoning Control Laboratory Monitoring Program of the Center for Disease Control (CDC). Based on the results of this monitoring mechanism, performance of the State Laboratory has been evaluated as acceptable or better. Yale-New Haven Hospital performed only macro tests and analyzed them using the atomic absorption method (*14*). The hospital laboratory meets state established quality control standards and also participates in the CDC proficiency blood lead testing program.

The reliance placed on blood lead measurement as the indicator of lead absorption in the New Haven study population made it necessary to assess the accuracy of the screening results, and particularly to assure that contamination of micro blood tests had not occurred. When micro test results were compared with macro test results (which were assumed to be accurate), after appropriate matching for age, race and date of sampling, there was no evidence to indicate any substantial inaccuracies of micro results or inconsistencies between results obtained by the two laboratories (*15*). For 410 children the micro test was followed within 60 days by a macro test. The mean difference in results was -7.1 μg/dl. This difference is not significant ($p > 0.05$) and could well have come about from an actual decrease or the error level inherent in the test.

The blood lead measurement is generally accepted as the best indicator of the external dose of lead, (*16*) where the external dose is the concentration of lead in environmental sources. Blood lead measurements are highly useful in the study of groups because the average blood lead level in a group reflects well the levels of current exposure and absorption of lead for the group as a whole (*17*).

Several authors have suggested that the distribution of blood lead levels for any relatively homog-

enous population closely follows a lognormal distribution (*18-20*). A variable is said to have a lognormal distribution if the logarithm of the variable is normally distributed. The skewed nature of the lognormal distribution makes the median (50th percentile) a more meaningful estimate of central tendency than the arithmetic mean. For the normal distribution, the best estimate of the median is $\bar{x}$, the simple arithmetic mean. For the lognormal distribution, the best estimate of the median (*21, 22*) is the geometric mean

$$GM = \text{antilog}\left[\sum_{i=1}^{n} (\log x_i/n)\right]$$

For this reason, the blood lead data presented in this report are geometric means.

Results

Geometric mean blood lead levels were stratified by the following factors: age, sex by age, race by age. From the 7912 intial tests available, analysis was based on tests on 7520 children in the study population for whom no measurements of environmental lead exposure had been obtained. Fifteen children were excluded because of missing information. Data for the remaining 377 children for whom environmental lead levels were determined have been reported upon separately (*23*). Exclusion of these 377 children had no effect on the results presented in this report.

Age

The initial analysis was a breakdown of geometric mean blood lead level of 7520 children by age in

Table 1. Geometric mean blood lead levels by age of children in New Haven, Conn., July 1, 1974–Feb. 28, 1977.

Age, months	Number of children	Blood lead level, μg/dl		
		GM[a]	GSD[b]	Range
1–12	663	25.5	1.4	8.0–97.1
13–24	1557	28.3	1.4	0.0–80.9
25–36	1198	28.7	1.4	0.0–75.0
37–48	1284	27.8	1.4	7.0–73.0
49–60	1365	27.4	1.4	3.0–74.0
61–72	1453	26.1	1.4	0.0–69.0
Total	7520	27.4	1.4	0.0–97.1

[a]GM = geometric mean blood lead level.

[b]$GSD = \text{antilog}\left\{\sum_{i=1}^{n} [\log (x_i) - \log (GM)]^2/(n-1)\right\}^{1/2}$

Table 2. Geometric mean blood lead levels in children aged 1-12 months in New Haven, Conn., July 1, 1974–Feb. 28, 1977.

Age, months	Number of children	Blood level, μg/dl		
		GM[a]	GSD[b]	Range
1	22	26.1	1.4	14.0–45.0
2	6	25.8	1.2	23.0–32.0
3	3	25.1	1.2	22.0–31.0
4	6	28.6	1.7	17.0–72.0
5	14	25.0	1.3	16.0–43.0
6	13	26.5	1.4	14.0–39.0
7	29	23.2	1.3	12.0–36.0
8	51	25.8	1.4	10.0–50.00
9	110	24.5	1.4	9.0–40.0
10	83	24.6	1.4	8.0–45.0
11	123	25.2	1.4	9.0–97.1
12	218	26.8	1.4	10.0–63.0
All ages	678	25.5	1.4	8.0–97.1

[a]GM = geometric mean blood lead level.

[b]$GSD = \text{antilog}\left\{\sum_{i=1}^{n} [\log (x_i) - \log (GM)]^2/(n-1)\right\}^{1/2}$

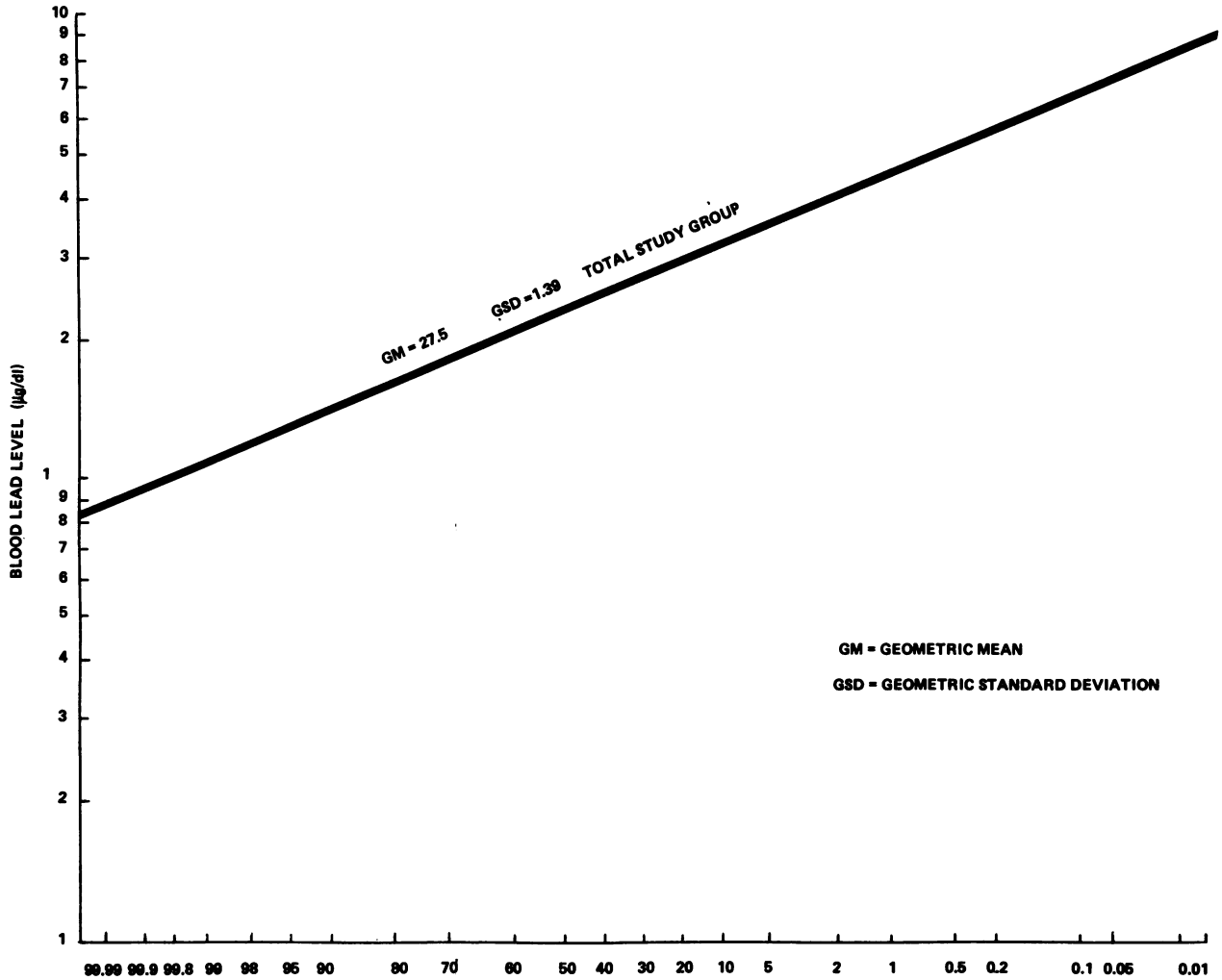

FIGURE 1. Estimated cumulative distribution of blood lead levels for the total sample having a geometric mean blood lead level of 27.5 µg/dl and geometric standard deviation of 1.39.

Table 3. Geometric mean blood lead levels by race and age of children in New Haven, Conn., July 1, 1974–Feb. 28, 1977.[a]

	Black			Hispanic			White			
	Blood lead, µg/dl			Blood lead, µg/dl			Blood lead, µg/dl			Differences significant
Age, months	GM	GSD	*N*	GM	GSD	*N*	GM	GSD	*N*	at 0.05 level
1–12	27.2	1.3	275	24.4	1.3	108	22.7	1.4	124	None
13–24	31.5	1.4	660	28.6	1.4	228	24.4	1.4	319	Black – white
25–36	30.9	1.4	531	28.5	1.4	186	25.3	1.5	264	None
37–48	29.2	1.4	628	29.6	1.4	169	24.3	1.4	298	None
49–60	29.2	1.3	704	28.2	1.3	189	23.6	1.4	361	Black – white
61–72	28.6	1.3	648	27.3	1.4	213	22.9	1.3	541	Black – white
Total	29.6	1.4	3446	27.4	1.4	1093	23.8	1.4	1907	Black – white

[a]GM = geometric mean blood lead level.

[b]$\text{GSD} = \text{antilog}\left\{\sum_{i=1}^{n} [\log(x_i) - \log(\text{GM})]^2/(n-1)\right\}^{1/2}$

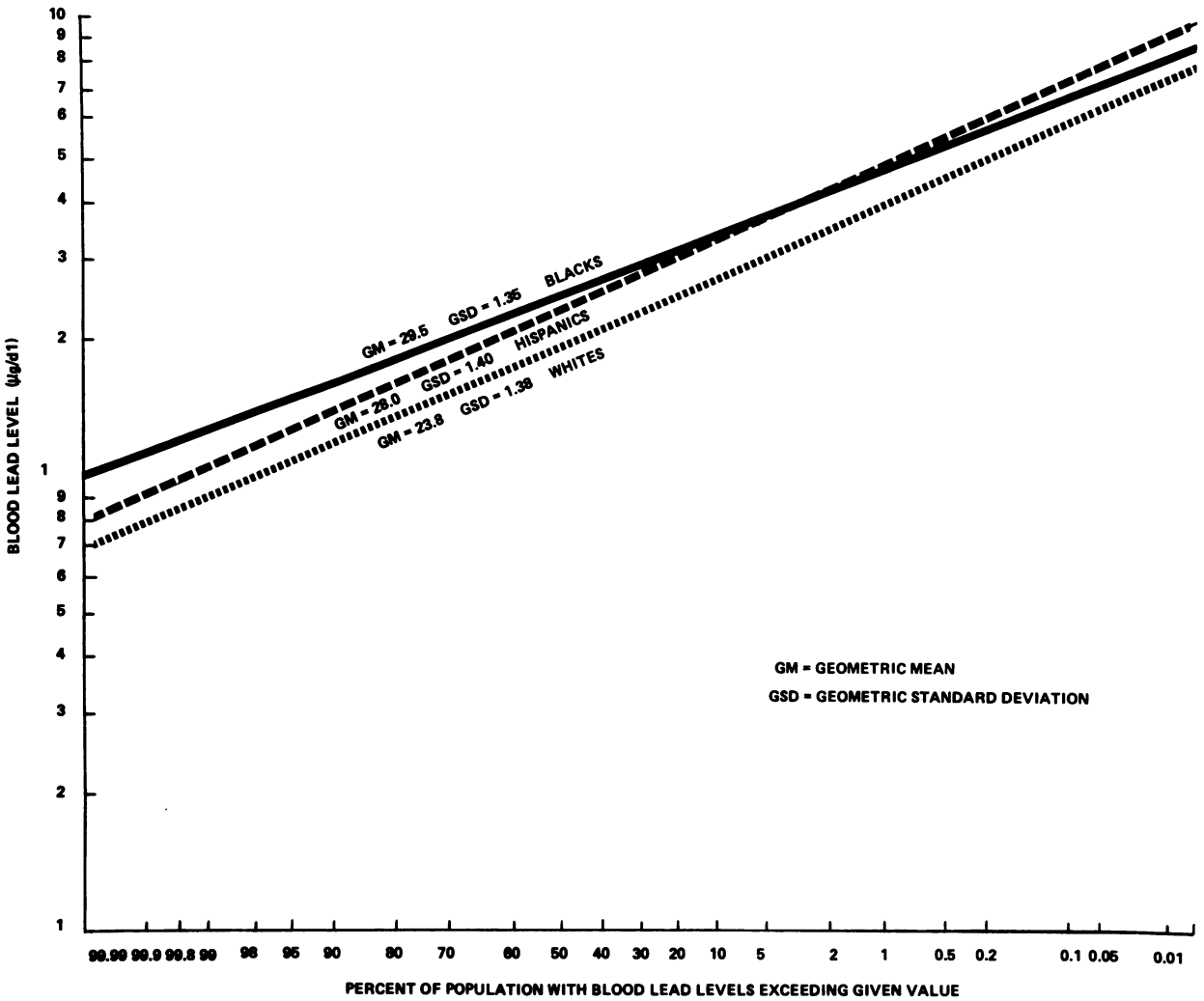

FIGURE 2. Estimated cumulative distribution of blood lead levels for blacks having a geometric mean of 29.5 µg/dl and geometric standard deviation of 1.35, for whites having a geometric mean of 23.8 µg/dl and geometric standard deviation of 1.38, for Hispanics having a geometric mean of 28.0 µg/dl and geometric standard deviation of 1.40.

months, at the time of test, as follows: 1-12; 13-24; 25-36; 37-48; 49-60; 61-72. It can be seen in Table 1 that the highest mean blood lead levels were between ages of about 25-36 months. The mean of the entire sample was 27.4 µg/dl with a standard deviation of 1.39. The range of blood lead levels by age category is considerable. Table 2 shows the distribution of geometric mean blood lead levels for the children in the 1-12 month age category by single month of age. Of the 678 children in the 1-12 month category, 614 were between 7-12 months old. The mean level for the 1-6 month old children was 26.1 µg/dl compared to a mean of 25.5 µg/dl for the 7-12 month old children, an insignificant difference. Figure 1 shows that 3.5% of the sample population had blood lead levels $\geq$ 50 µg/dl and 39% had levels $\geq$ 30 µg/dl.

Sex by Age

Sex was recorded in 7462 of 7520 cases. When mean blood lead levels were compared by age and sex, the levels of the two sexes did not differ significantly. Therefore, results for both sexes were combined in subsequent analyses.

Race by Age

Genetic characteristics were not defined in this study, but family self-definition was used to assign each child to a racial/ethnic/cultural category. Although there were a small number of people who did not classify themselves as belonging to one of the three major racial groups in New Haven (black, white, Hispanic), the "other" category was too small to be

included in analysis. Race data were missing in about 14% of subjects. Table 3 gives the results of geometric mean blood lead levels by race and age. For all ages the lead levels of whites were substantially lower than those of blacks and Hispanics. Until age 37-48 months, levels for Hispanics were lower than for blacks, but starting with that age, the levels of these two groups were similar. Between the ages of 18-42 months, the geometric mean blood lead level for black children in New Haven was greater than 30 μg/dl.

Comment

A geometric mean (i.e., a median) blood level of 27.4 μg Pb/dl in an 80% sample of the entire population of New Haven children aged 1-72 months shows that a good deal of lead was available to them. Much of the lead found in these children's blood can be presumed to have come from the external physical environment rather than from food or beverages. This inference is supported by the peak values in children between about 18 and 42 months of age, when normal mouthing behavior would be expected to maximize the oral intake of nonfood materials. Contamination of these materials with even small amounts of lead would produce the results observed. The source of the high levels of lead found in the blood of very young infants is less clear. Prenatal exposure cannot be ruled out. The implications of these high blood lead levels in terms of effect on the vulnerable developing nervous system are cause for concern.

The substantial differences in mean blood lead levels among different groups classified by racial category indicate the importance of this variable. Figure 2 shows the estimated cumulative distributions of blood lead levels for black, white and Hispanic children. It can be seen that 50% of the black children, 43% of the Hispanic children and 23% of white children had blood lead levels ≥ 30 μg Pb/dl. This leaves no room for complacency. Similar population surveys in other areas are needed to place the New Haven results in a broader prospective. A random sample of adults should be included. The design and implementation of prevention and treatment strategies for childhood lead poisoning will require ongoing surveillance of populations whose demographic characteristics are clearly defined.

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REFERENCES

1. Chisolm, J. J., Jr. Is lead poisoning still a problem? Clin. Chem 23 (2): 252-255 (1977).
2. Gilsinn, J. F. Estimates of the Nature and Extent of Lead Paint Poisoning in the United States. NBS Technical Note 746, U.S. Dept. of Commerce, National Bureau of Standards, Washington, D.C., 1972.
3. Center for Disease Control. Morbidity and Mortality Weekly Reports, 30 (No. 5): 63 (Feb. 13, 1981).
4. EPA. Air Quality Criteria for Lead, Office of Research and Development, U.S. Environmental Protection Agency, EPA-600/8-77-017, Dec. 1977.
5. Christian, J. R., Celewycz, B. S., and Andelman, S. L. A Three-year study of lead poisoning in Chicago. Am. J. Publ. Health 54: 1241-1245 (1964).
6. Center for Disease Control. Increased Lead Absorption and Lead Poisoning in Young Children, U.S. Dept. of Health, Education and Welfare, PHS, March 1975.
7. Jacobnizer, H. Lead poisoning in childhood: epidemiology, manifestations, and prevention. Clin. Ped. 5: 277-286 (1966).
8. Chisolm, J. J., Jr., Lead poisoning. In: Pediatrics, 15th ed. H. L. Barnett, Ed., Appleton-Century Crofts, 1972, pp. 540-548.
9. Rapoport, M., and Rubin, M. I. Lead poisoning: a clinical and experimental study of the factors influencing the seasonal incidence in children. Am. J. Dis. Child. 61: 245-255 (1941).
10. Stark, A. D., Quah, R. F., Meigs, J. W., and DeLouise, E. R. Season as a factor in variability of blood-lead levels in children. Conn. Med. 44(7): 410-412 (1980).
11. Stark, A. D., Meigs, J. W., Fitch, R. A., and DeLouise, E. R. Family operational co-factors in the epidemiology of childhood lead poisoning. Arch. Environ. Health. 33: 222-225 (1978).
12. Stokinger, H. E. Recent History of Lead Exposure in the U.S. Industry, Symposium of Environmental Lead Contamination. Bureau of Community Environmental Management, PHS Publ. No. 1440, Dec. 1975.
13. Olsen, E. D., and Jatlow, P. I. An improved Delves' cup atomic absorption procedure for determination of lead in blood and urine. Clin. Chem. 18: 1312-1372 (1972).
14. Zinterhofer, L. J. M., Jatlow, P. I. and Fappiano, A. Atomic absorption determination of lead in blood and urine in the presence of EDTA. J. Lab. Clin. Med. 78: 664-674 (1971).
15. Quah, R. F., Stark, A. D., Meigs, J. W., and DeLouise, E. R. Micro determination of blood-lead; reliability for mass screening of children. Conn. Med. 44(4): 210-213 (1980).
16. Chisolm, J. J., Jr., Barrett, M. B., and Harrison, H. V. Indicators of internal dose of lead in relation to derangement in heme synthesis. Johns Hopkins Med. J. 137: 6-12 (1975).
17. Chisolm J. J., Jr. Current status of lead exposure and poisoning in children. Southern Med. J. 69: 529-531 (1976).
18. Yankel, A. J., VonLindern, I., and Walter, S. D. The Silver Valley lead study. The relationship between childhood blood-lead levels and environmental exposure. J. Air Pollut. Control Assoc. 27: 763-767 (1977).
19. Tepper, L. B. and Levin, L. S. A survey of air and population lead levels in selected American communities. Environ. Qual. Safety (Suppl. II: Lead) 254 (1975).
20. Azar, A., Snec, R. D. and Habibi, K. An epidemiologic approach to community air lead exposure using personal air samples. Environ. Qual. Safety (Suppl II: Lead) 254 (1975).
21. Aitchison, J., and Brown, J. A. C. The Lognormal Distribution, Cambridge Univ. Press, London, 1966.
22. Johnson, N. L., and Kotz, S. Continuous Univariate Distributions 1, Houghton Mifflin, Boston, 1970.
23. Stark, A. D., Quah, R. F., Meigs, J. W., and DeLouise, E. R. The relationship of environmental lead to blood-lead levels in children. Environ. Res. In press.